

1993

Sulfide as a toxicant in aquatic habitats

Bagarinao, Teodora U.

Aquaculture Department, Southeast Asian Fisheries Development Center

Bagarinao, T. U. (1993). Sulfide as a toxicant in aquatic habitats. SEAFDEC Asian Aquaculture, 15(3), 2-4.

<http://hdl.handle.net/10862/1791>

Downloaded from <http://repository.seafdec.org.ph>, SEAFDEC/AQD's Institutional Repository

Sulfide as a Toxicant in Aquatic Habitats*

T.U. Bagarinao, Scientist, SEAFDEC/AQD

Hydrogen sulfide is more than just a disagreeable odor from a stagnant marsh: it is a serious menace to all aerobic organisms. Sulfide has a wide variety of adverse effects, the major one being the inhibition of cytochrome *c* oxidase, the terminal enzyme in aerobic respiration located in the inner membrane of mitochondria. As a toxicant that occurs widely in aquatic (particularly marine) habitats, sulfide may influence the health, survival, productivity, and distribution of various organisms. The effects of sulfide on mammals are well-known and are discussed in some detail here because at the biological level they are the same as in plants, invertebrates, and fishes.

Dose- and pH-dependent toxicity

Sulfide toxicity is dose-dependent for any one species and varies with the particular biological system (e.g., whole organism, isolated mitochondria or enzyme preparation) under study. Nanomolar sulfide concentrations are sufficient to inhibit cytochrome *c* oxidase, while micromolar concentrations are toxic at the whole-animal level. Toxicity depends on the balance between the diffusion rate of sulfide towards the enzyme, and the rate at which sulfide is removed or detoxified by agents external

*Source: *Aquatic Toxicology* 24:21-62, 1992.

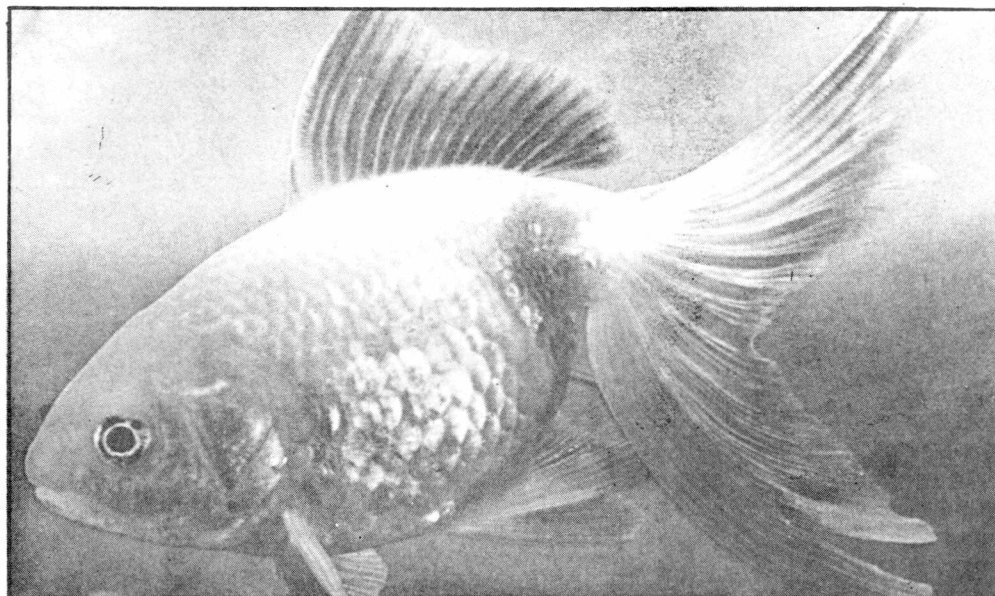
to the enzyme. Some species are more tolerant to sulfide than others and utilize various means of coping with sulfide toxicity, sometimes several mechanisms in concert.

The concentration of sulfide that is toxic depends on pH. In seawater of typical pH 8.3, about 6% is H_2S ; in sediments of typical pH 7.0, about 50% is H_2S . The rate of diffusion of sulfide into cells is directly proportional to the concentration of H_2S in the external solution. H_2S freely crosses membranes, while the HS^- anion may be electrically excluded. However, HS^- anions may also contribute to toxicity at high sulfide concentrations. In many organisms, sulfide toxicity is modulated by pH.

Toxicity to mammals

The toxic effects of sulfide are best understood in mammals and are generally similar in aquatic organisms. Sulfide is an industrial health hazard for people working in oil wells and refineries, kraft paper mills, tanneries, sewers, manure pits, fishing fleets, and hot-spring reservoirs. At the physiological level, sulfide has two major effects on mammals: (1) local inflammation and irritation of moist membranes including the eye and respiratory tract, and (2) cardiac arrest due to paralysis of the respiratory centers of the brain. Prolonged exposure to as low as 50 ppm H_2S in air causes inflammation, dryness,

Freshwater fishes such as the goldfish are less tolerant of sulfide than marine fishes such as the California killifish that lives in salt marshes. Sulfide toxicity increases with temperature, decreases with pH and oxygen concentration, and varies with fish.



hoarseness, cough, bronchitis, pneumonia, and at concentrations exceeding 250 ppm in air, also lung edema. Lung edema is the single most notable lesion in human cases of sulfide inhalation poisoning. Intravenous or intraperitoneal administration of sodium sulfide has similar effects on respiration, except lung edema.

Adverse effects of sulfide on the nervous system include headache, lightheadedness, sleep disturbances, drowsiness, fatigue, spasms, disturbed equilibrium, convulsions, agitation, and in severe cases, deep coma, nerve paralysis, unconsciousness, and death. Like cyanide, sulfide also initially causes rapid breathing, then cessation of breathing and death. About 700 ppm H_2S in air is rapidly fatal to humans; very low concentrations (0.02-0.13 ppm) can be smelled and give some warning, but 150 ppm in air paralyzes the olfactory nerve.

The effects of sulfide at the whole-organism level have their basis at the cellular and molecular level. The *in vivo* biochemical effects of sulfide in mammals include: (1) inhibition of cytochrome c oxidase and oxidative phosphorylation, resulting in tissue hypoxia and ATP depletion; (2) metabolic impairments due to changes in enzyme activities, metabolites and cofactors; (3) production of reactive radicals and alteration of membrane permeability, causing edema and organ-specific functional disorders; (4) neurotoxicity; and (5) changes in blood proteins.

About 20 other enzymes are inhibited by sulfide—including superoxide dismutase, catalase, and glutathione peroxidase that act against cellular injury caused by reactive oxygen species. Inhibition of these enzymes as well as the stimulation of xanthine oxidase by sulfide allow cellular injury to proceed unchecked.

Neurotoxicity and particularly the arrest of central respiratory drive during sulfide poisoning in mammals may be due to the: (1) extremely rapid alterations in amino acid neurotransmitter levels in the brainstem, (2) inhibition of monoamine oxidase, and (3) disruption of action potentials and sodium channel function. Moreover, sulfide at high concentrations *in vitro* causes the formation of sulfhemoglobin and sulfmyoglobin, which can no longer transport oxygen. However, mammals and fishes poisoned by sulfide rarely contain sulfhemoglobin in the bloodstream, and impairment of oxygen transport is not involved in acute sulfide poisoning.

Toxicity to plants

Sulfide is a causal factor in 12 of 27 physiological disorders of rice, and is the primary cause of straighthead disease and mild sulfide disease. Sulfide inhibits respiration, oxygen release, and nutrient uptake by rice roots. Disease-resistant cultivars show higher tolerance to sulfide. Likewise, sulfide inhibits the growth of the salt marsh plants *Puccinella maritima*, *Atriplex patula* and *Festuca rubra*, but not of *Salicornia europaea*, which is able to establish and grow in areas of the lower marsh from which the others are excluded by sulfide. *Spartina alterniflora*, the cordgrass that dominates the salt marshes of the US east coast, takes up sulfide without acute toxicity effects over a long growing season.

Sulfide has complex effects on photosynthesis, cell division, respiration, assimilation and fermentative ability in cyanobacteria and unicellular algae. Some species and strains of algae could multiply in the presence of 250-500 μM sulfide, whereas others are inhibited in 30-60 μM sulfide. Photosynthesis in some cyanobacteria decreases in the presence of sulfide, with 50% inhibition at 100 μM sulfide in non-heterocystous species.

Toxicity to macro-invertebrates

Aquatic invertebrates have been studied in terms of the tolerance levels and adaptations to sulfide. In tolerance studies, sulfide exposure was coupled with hypoxia or anoxia and the effects compared with those of hypoxia or anoxia alone. In all cases, sulfide was shown to worsen the effects of hypoxia and anoxia. Various marine worms including the lugworm *Arenicola marina* survive 2-5 days of exposure to 10 mM sulfide. The burrowing intertidal worm *Cirriiformia tentaculata* survives 10 days under anoxia and 5 days under anoxia plus 200 μM sulfide. Similarly, the tube-dwelling worm *Nereis diversicolor*, found usually in silty sediments in the innermost parts of estuaries and fjords, is more tolerant to anoxia plus 180 μM sulfide than *N. virens*, which occurs in oxidized sand bottoms. The priapulid worm *Halicryptus spinulosus* is able to survive exposure to 200 μM sulfide for at least 40 days.

The infaunal sea star *Ctenodiscus crispatus* from muddy bottoms survives hypoxia plus 1.5 μM sulfide for 10

The role of sulfide in mass kills of fish, shrimps, and other animals in brackish-water earthen ponds, lakes, and sea cages should be determined.

days, while *Asterias vulgaris* and *A. forbesi* from the rocky intertidal survive only 4-5 days under the same conditions. Survival of the coot clam *Mulinia lateralis* during anoxia plus sulfide is much lower than under anoxia alone, decreases with sulfide concentration (200 μ M to 2.68 mM) and temperature (10°C and 20°C), and increases with the size of the clam. Of two mudflat clams, *Macoma nasuta* is more tolerant to sulfide than *M. secta*, and both show lower tolerance to anoxia in the presence of sulfide.

Compared with other benthic invertebrates, crustaceans have low tolerance to hypoxia and sulfide. The heart rate of the vent crab *Bythograea thermydron* is unaffected by >1 mM sulfide, while those of three shallow-water crab species are severely affected when sulfide concentrations reach 300 μ M. *Pachygrapsus crassipes*, which burrows in sulfide-rich sediments in salt marshes, is less affected by sulfide than *Cancer antennarius*, which lives on sand and rocks, and *Portunus zantusii*, which is free-swimming. In continuous-flow bioassays of eight species of freshwater crustaceans and insects, the 96-hour LC_{50} H_2S (lethal concentration for 50% of the animals) range from 0.6 μ M in *Baetis* to 31 μ M in *Asellus*.

Toxicity to freshwater fishes

Ironically, the toxic effects of sulfide were first studied in freshwater habitats where less sulfide is produced than in comparable marine habitats. These studies were conducted initially in relation to pollution of lakes and streams by sewage and kraft mill effluents, and later in recognition of sulfide itself as a factor in fishery and aquaculture production. The documented effects of sulfide on freshwater fishes include: (1) enhanced survival and growth at low sulfide concentrations between 0.02 and 0.4 μ M H_2S , attributed to the antibiotic effect of sulfide; (2) reduced survival and growth at sulfide concentrations greater than 0.45 μ M H_2S ; (3) lower swimming endurance; (4) tissue irritation and death; (5) lower food consumption and conversion; (6) inhibited spawning behavior and reduced egg production; and (7) lower survival of eggs and smaller size and higher incidence of deformities in newly hatched larvae. A 30-day exposure of sexually maturing common carp *Cyprinus carpio* to a sublethal concentration of 280 μ M total sulfide (about 28 μ M H_2S) causes a gradual decrease in gonad size due to liver malfunction.

Among the juveniles of eight species of freshwater fishes, the 96-hour LC_{50} H_2S concentrations vary from 0.1 μ M at 25°C in goldfish *Carassius auratus* to 23 μ M at 6.5°C in the fathead minnow *Pimephales promelas*. The fry stage is up to three times more sensitive to sulfide than the juveniles. Sulfide toxicity increases as temperature rises, as oxygen concentration falls, and as pH decreases.

Toxicity to marine fishes

Many species of marine fishes occur in areas with low to high levels of sulfide. In field tests in cages near pulp and paper mills in Port Angeles (Washington) harbor, the mortality of juvenile salmon occurred at H_2S concentrations of 4 μ M and greater. The codlet *Bregmaceros nectabanus* spends 10-11 hours in the anoxic, sulfide-containing zone of the Cariaco Trench off Venezuela during vertical migration over a depth range of 800 meters. Many deep-sea fishes have been observed at the hydrothermal vents, and three endemic genera (*Thermarces*, *Bythites*, and *Thermobiotes*) seem to have some sort of obligate relationship with the vents in both Pacific and Atlantic Oceans. Several species of fish have also been obtained from trawls in the sulfide-rich hydrocarbon seep 600 meters deep off Louisiana. Groupers, cottonwicks and red snappers swim freely in and out of the sulfidic stream at the East Flower Garden brine seep 72 m deep in the Gulf of Mexico.

Sulfide tolerance differs among marine fishes from a salt marsh, an enclosed bay and the open coast, being high, intermediate and low, consistent with the relative sulfide levels that may be encountered in these habitats. The California killifish *Fundulus parvipinnis*, a salt marsh resident, is highly tolerant of sulfide, the 96-hour LC_{50} being 700 μ M and the 8-hour LC_{50} being 5 mM total sulfide (or 42 and 300 μ M H_2S). The speckled sanddab *Citharichthys stigmaeus* from the open coast is intolerant of sulfide, dying within 2 hours at a constant 200 μ M total sulfide. The killifish tolerates sulfide levels 100 - 1000 x greater than those that inhibit cytochrome c oxidase. This is because killifish mitochondria can oxidize (10 - 20 μ m) sulfide to thiosulfate and remove it before it reaches the enzyme.

The role of sulfide in mass kills of fish, shrimps and other animals in brackishwater earthen ponds, lakes and sea cages should be determined.

First African Fisheries Congress

1 - 5 August 1994

Nairobi, Kenya

The Fisheries Society of Africa (FISA), a member of the World Council of Fisheries Societies, is organizing the First African Fisheries Congress in Nairobi, Kenya, 1-5 August, 1994.

FISA and the Organizing Committee of the African Congress invite interested parties to a roundtable discussion on South-South Cooperation in Research and Development. For further information, write, call or fax: Fisheries Society of Africa, Department of Zoology, University of Nairobi, P.O. Box 30197, Nairobi, Kenya, Tel. 442316 or 442121 Ext. 536. Fax: 254-2-336885.